

In the Claims:

Please amend the claims by deleting the text shown as strikethrough and adding the text shown in underline.

1. (Currently amended) A method of monitoring and modulating a disease-associated activatory process, wherein the activatory process comprises activation of cytokinsecretion, induction of direct cell-bound signals, or transmission of signals regulating proliferation, differentiation, and/or senescence, the method comprising determining and influencing the amount or activity of caspase-10 or caspase-10 isoforms in a cell or an organism, wherein the activatory process is triggered which is affected by non-apoptosis signals emanating from death receptors or non-apoptosis signals emanating from non-death receptor members of the TNF receptor family regardless of the disease being treated.
2. (Original) The method of claim 1 wherein the activatory process is triggered by receptor-crosslinking.
3. (Currently amended) The method of claim 1 or 2, wherein the activatory process is triggered amount or activity of caspase-10 or caspase-10 isoforms is affected by non-apoptosis signals emanating from death receptors selected from TRAIL-R1, TRAIL-R2, CD95, TNF-K1 (pSS TNF-R), TRAMD, DR6 and combinations thereof.
4. (Currently amended) The method of claims 1 or 2, wherein the activatory process is triggered amount or activity of caspase-10 or caspase-10 isoforms is affected by signals emanating from non-death receptor members of the TNF receptor family and/or from death receptor members of the TNF receptor family or members of the TLR receptor family.
5. (Previously presented) The method of any one of claims 1 or 2, wherein the disease is selected from hyperproliferative, inflammatory and auto-immune diseases.

6. (Original) The method of claim 5, wherein the disease is an inflammatory disease selected from skin inflammatory diseases and septic shock.
7. (Original) The method of claim 5, wherein the disease is a hyperproliferative disease selected from tumors.
8. (Original) The method of claim 5, wherein the disease is an auto-immune disease.
9. (Previously presented) The method of any one of claims 1 or 2 comprising monitoring the presence, amount, localization or activity of caspase-10 or caspase-10 isoforms in a sample.
10. (Original) The method of claim 9, wherein caspase-10 or caspase-10 isoforms are determined on the nucleic acid level.
11. (Original) The method of claim 9, wherein caspase-10 or caspase-10 isoforms are determined on the protein level.
12. (Previously presented) The method of any one of claims 1 or 2 comprising modulating the amount or activity of caspase-10 or caspase-10 isoforms in a cell or an organism.
13. (Previously presented) The method of claim 12, wherein the amount or activity of caspase-10 or caspase-10 isoforms is modulated on the nucleic acid level.
14. (Previously presented) The method of claim 12, wherein the amount or activity of caspase-10 or caspase-10 isoforms is modulated on the protein level.
15. (Cancelled) A method of identifying or characterizing compounds for the modulation of a disease-associated activatory processes comprising determining if a test compound is capable of influencing the activity of caspase-10 or caspase-10 isoforms, wherein the activatory processes are triggered by non-apoptosis signals emanating from death receptors or non-apoptosis signals emanating from non-death receptor members of the TNF receptor family.